

**Your Premier Information Resource for Lone Atrial Fibrillation  
Publisher: Hans R. Larsen MSc ChE**

## **VIRTUAL LAF CONFERENCE**

Proceedings of 48th Session  
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### **SUBJECT: LAF and Insulin**

The results of LAFS-11 have generated some initial discussion on the Bulletin Board, so it's been imported to the Conference Room for a more in depth review.

Those results point toward body shape (v. size) as a more significant determinant of LAF (v. AF). This information in conjunction with the results of LAFS-5 appears to implicate insulin sensitivity (in at least a subset of those with LAF) and potassium imbalance as vital to its pathogenesis.

First of all, I fully admit that this survey was not strictly random. Willingness or not to complete it was in the hands of those receiving it, i.e., those with email addresses in Hans' massive database.

Some may not have felt comfortable divulging anthropometric and lab data, despite assurances of anonymity and/or confidentiality. This may have resulted in a bias toward the thin and away from the not so thin. I personally don't think this was significant, but what do others think.

Secondly, I would suggest that there maybe no such thing as pure adrenergic LAF. Given the anthropometric data of LAFS-11, it would appear that most are taller with less central obesity than their normal counterparts. Enhanced insulin sensitivity is an obvious explanation that fits with the limited lab data available. If one accepts this, then a whole new way of looking at LAF arises. Remember that insulin sensitivity/resistance, autonomic balance, lipoprotein profile and inflammation are all inextricably entwined for everyone, not just LAFers. In view of this connection it would seem that adrenergic LAF does not indicate increased sympathetic activity v. the normal population, just less parasympathetic tone than VMAFers. Their episodes are triggered by stress, and this translates to a greater assault on their potassium balance than for VMAFers, since the role of aldosterone is greater in adrenergics. Those in the adrenergic category seem to be shrinking in number. Perhaps many former adrenergic LAFers are recognizing that many of their episodes are vagal in nature, hence the growing number in the mixed category. Therefore, IMHO stress related LAF should shed its tarnished image and be clearly separated from AF with organic heart disease, always accompanied by increased sympathetic tone v. the normal population.

Thirdly, many LAFers do not have a significant problem with hypoglycemia (50% in LAFS-5). This does not mean that insulin is not integral to their LAF. It's not just about hypoglycemia. It is also about the windows the hypoglycemia connection open to understanding the pathogenesis of LAF (IS/IR, autonomic balance, lipoprotein profile and inflammation). Are there some LAFers with insulin resistance? Obviously it depends on your definition and by most definitions there are. However, could some of this insulin resistant LAF represent an early manifestation of more traditional AF in which discernible organic heart disease has not yet appeared? This is not meant to alarm anyone only to help in our understanding. We are what we are and knowledge is always more useful than ignorance.

In a very recent article appearing in the **European Heart Journal**, entitled  
*"Left Atrial Volume Predicts Cardiovascular Events In Patients Originally Diagnosed With Lone Atrial Fibrillation: Three-Decade Follow-Up"*

<http://eurheartj.oxfordjournals.org/cgi/content/full/26/23/2556>

some interesting data on prognosis was presented.

"The observed mortality at 25 years after onset of lone AF was lower (15.9%) than expected (32.5%) based on age- and sex-specific rates from the general Minnesota white population life tables."

And this study included LAFers with left atrial volume > 32 mL/m<sup>2</sup>. All strokes in that study occurred in those with a left atrial volume greater than 32 mL/m<sup>2</sup>. Could this stroke prone subset be different anthropometrically?

I've stated that LAFers may live longer than their normal counterparts because they may have less insulin and IGF-1 onboard. Additionally HDL levels may be elevated in LAFers, thereby contributing additional protection against CV disease and enhancing longevity.

Please feel free to comment on the above. Dissenting views are most welcome. Apologies in advance to any LAFers offended by these speculative remarks. We must all remember that there are many diseases worse than LAF and that LAF can be a strong motivator to improving lifestyle.

Hopefully ensuing discussion will lead to support for another survey looking at total cholesterol, HDL cholesterol, triglycerides, white blood cell count, family history, birth weight and length of gestation, ?premature at birth. There is some evidence that the incidence of diabetes is greater in babies born premature and/or small for dates babies. Perhaps LAFers are more likely to be bigger at birth and/or born after their due date (postmature).

## **PC**

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PC, I'm glad this subject made it to a Conference Room. After two years of gradually eliminating other possible sources of my VMAF, I have recently become more suspicious of insulin and its multiple effects in the body. In my case, I think a key fact is that insulin is critical for storing Mg in cells. This may hold the key for many on this board who test at the low end of normal Mg and have difficulty raising their Mg results, even after years of substantial supplementation.

I've just finished reading what Dr. Ron Rosedale (The Rosedale Diet) has to say about insulin sensitivity as well as leptin sensitivity. There are implications of retaining sodium and calcium as well as deficiencies of Mg and, ultimately, K, which are all issues LAFers need to be concerned about. There's also a slew of other symptoms that may apply to many of us.

I know in my family, my father and at least 4 of his 6 brothers were all diagnosed with afib and, eventually, Type 2 diabetes. His youngest two brothers are also diabetic, but no afib yet. One of my brothers and many of my cousins are pre-diabetic and I, at 56 and the oldest of that next generation, developed LAF two years ago. Although my glucose tests normal, I have not yet been tested to determine insulin sensitivity, even though I have many of the symptoms. At least one of my cousins has had afib episodes and I suspect that others are soon to follow based on discussions I've had with them about health.

Remember, however, that one need not be diabetic to have issues with insulin resistance.

I have been helped some by Mg and K supplementation, but it's like I can barely hold a minimum level to hold off PAC's and PVC's. Both get worse in a hurry if I stop my supplements. But why can't I seem to build up and maintain higher levels of Mg and K? I think it has to do with insulin resistance. This is the direction I'm now taking my search.

The Paleolithic diet, which has helped so many on this board, is basically a diet that would restrict the sugar and simple carbs that would tend to spike insulin and leptin, eventually causing resistance. And, it appears that both insulin and leptin sensitivity can be substantially corrected with diet.

Perhaps the biggest result from eating a diet similar to the Paleolithic diet is, in fact, allowing insulin to correctly store Mg where we need it in quantities sufficient to maintain NSR and minimize PAC's and PVC's.

## **TerryM**

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Just want to post a link to Ron Rosedale's wonderful 4 part insulin article, "Insulin and its metabolic effects", invaluable for understanding the whole insulin situation. This is the Rosedale article Terry mentions above.

<http://www.mercola.com/2001/jul/14/insulin.htm>

**PeggyM**

Hi PC

If you go to this site referenced by Todd on the regular board:

<http://www.magnesiumforlife.com/mineraltherapy.shtml>

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You will notice that in their before/after cellular electrolyte profile (in a table in the middle of the page) that the Ca<sup>+</sup> is very high before & within range after. I don't have access to my own data, as I'm traveling, however from my memory, my test mirrored the before test.

Some place, perhaps in the written Exatest material, I remember that this (high Ca<sup>+</sup>) was indicative of insulin resistance. I'm wondering if the 7 people who submitted Exatest results also had a similar profile. If so, this might indicate that these low Mg people have a tendency to hypoglycemia because of insulin resistance, not enhanced sensitivity to insulin.

BTW the results are impressive. I'm thinking of seeing if Mag chloride ice melter has any deleterious components, or if you could just soak in it. It is pretty inexpensive in bulk.

**George**

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Aloha Terry, Peggy and George,

Thank you all for elevating this topic from its previous nondiscussion status.

It seems to me that there is a growing controversy about the role of insulin resistance (v. insulin sensitivity) in the genesis of lone atrial fibrillation (LAF).

I think all your posts are pretty correct as far as the role of insulin resistance in the genesis of AF. However, it is my personal belief that there are two kinds of LAF. One due to hypoglycemia associated with insulin resistance and the other with insulin sensitivity. Based on the results of all the previous surveys (LAFS 1-11), the vast majority of those responding appear to have LAF associated with insulin sensitivity.

Insulin resistance is associated with prediabetes and diabetes, present in 15% and 7% of the American population respectively, at least according to the American Diabetic Association. Previous LAF surveys in 2001 and 2003 revealed diabetes to be present in 0 of 50 in 2001 and in 2 of 202 in 2003, one of whom had permanent LAF. Yet 50% of the respondents in 2001 admitted to hypoglycemia or symptoms of hypoglycemia and 25% in 2003 admitted to hypoglycemia alone. This is not exactly a ringing endorsement of insulin resistance among LAFers.

Insulin resistance, increased sympathetic tone, overweight/obesity, bad lipoprotein profile (high LDL and low HDL) and chronic inflammation are all directly related and inextricably entwined. Anthropometric data from LAFS - 11 does not support the notion that insulin resistance is present to any degree amongst LAFers.

Undoubtedly there are some insulin resistant LAFers that are without discernible organic heart disease and there is no way to date to differentiate them from those with LAF associated with insulin sensitivity.

Based on the results of LAFS-11, my proposal is to enlist HDL/triglycerides, white blood cell count, measures of autonomic tone, age at onset, blood pressure and anthropometric data to do this.

The reason for this distinction within LAF is to more sharply delineate optimal therapeutic regimens and prognosis.

Those with LAF associated with insulin resistance may be more susceptible to stroke. Please visit <http://eurheartj.oxfordjournals.org/cgi/content/full/26/23/2556>

for an interesting article related to this view. I believe, and this article supports, the notion that those with LAF associated with insulin resistance are more likely to develop diastolic dysfunction and subsequent atrial enlargement.

You might ask, "What about adrenergic LAF"? Don't they have greater sympathetic tone?

IMHO ALL those with insulin sensitive LAF have greater vagal tone than the general population. It's just that VMAFers have more than adrenergics. Accordingly, the latter require more input from potassium imbalance to trigger an episode. Remember that AERP shortening (vagal tone causes more of this than sympathetic tone) and PAC couplets appear to rule the roost on triggering LAF episodes. Hans would be the prototypical adrenergic LAFer. Adrenergics may have a somewhat sharper drop in their blood potassium levels (v. VMAFers) due to the simultaneous action of insulin, catecholamines and aldosterone during stress.

If adrenergics truly had more sympathetic tone than the general population, then there would be many overweight adrenergic and mixed LAFers. LAFS-11 did not reveal this. LAFers are thinner than not only the general population but also the insulin resistant. In general overweight/obesity cannot occur in the absence of increased sympathetic tone and vice versa.

VMAFers initially have episodes only at night, when vagal tone is highest both on a diurnal basis and on a positional basis. In addition blood potassium has a diurnal nadir at midnight. If you're a little magnesium deficient, then the situation is a little worse. Leakage of intracellular potassium is commensurately increased (v. a more acute drop during the day for adrenergics).

As episodes increase in duration and frequency the atria become slightly dilated and inflammation gradually increases. Both are effects of LAF (associated with insulin sensitivity) not causes of it. The stretching causes PACs. VMAF episodes then begin to occur during daytime, because less potassium shortfall is required to trigger the PACs for an episode. Pure adrenergics also gradually become mixed, as episodes can eventually be triggered by atrial stretching due to postural changes. For both once sufficient atrial stretching has transpired potassium imbalance becomes less required for PAC production.

This is undoubtedly why some receive benefit from potassium supplementation and some do not. It all depends on where on the spectrum you lie.

Stretching is definitely at the heart of AF and LAF. PVs in both are more dilated than normal. And the R and L superior PVs are more dilated than the R and L inferior PVs. And that's precisely where the PACs originate (superior PVs). During an episode of AF after the mitral valve has slammed shut during a ventricular contraction and the left atrium has been incompletely emptied of blood, the resulting wave of blood would deliver more mechanical stress directly opposite the mitral valve, which is where the superior PVs are located. The inferior PVs would only get a glancing blow. This surmise certainly would predict greater mechanical stress on the superior PVs than the inferior PVs.

The validity of this mechanistic approach is underscored by the fact most LAFers have normal sized atria in all dimensions except the longitudinal direction (top to bottom). This is not generally appreciated on most echocardiograms. LAFers that subsequently develop ventricular diastolic dysfunction (unrelated to LAF) then go on to develop additional atrial enlargement in the other dimensions (not caused by a unidirectional wave of blood but by "backup in the plumbing"). For them the mechanical stress is more equally distributed throughout the atria. Once 32mL/m<sup>2</sup> left atrial volume is exceeded the risk for stroke rises quickly.

Please feel free to challenge any of the above. It would be most welcome in this otherwise anemic CR. Additional references are available for much of it.

**PC**

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PC,

As usual, I'm just throwing in contrary thoughts. In my own case, even though my own data is in line with a (relatively) low w/h, w/height ratios, good HDL/LDL, low triglycerides & etc. and I am relatively lower body overweight -- my thighs are like tree trunks from my football days, 18 months ago I had the Exatest profile with low Mg & high Ca. This led me to explore my own blood glucose status in detail with a glucometer.

From following my blood glucose over the course of a year, I think that I am neither hypo or hyper glycemc. My hemoglobin A1C, my reading this fall was 5.1. From Jackie's post: "However Ron Rosedale MD says that 5.4 is optimal, 5.6-5.8 is acceptable, 5.9-6.9 is high and 7.0 is a diabetes risk. My last values were 5.0 and then up to 5.5." [http://www.afibbers.net/forum/read.php?f=4&i=256&t=166#reply\\_256](http://www.afibbers.net/forum/read.php?f=4&i=256&t=166#reply_256)

The reference range for normals is 4.5 - 6.0 (from memory).

In simple terms, the A1C is an indication of your average blood glucose over a three-month period. Here is the correlation between A1C & glucose

A1C(%) Mean blood sugar (mg/dl)

4.4	79
4.5	82.5
4.6	86
4.7	89.5
4.8	93
4.9	96.5
5	100
5.1	103.5
5.2	107
5.3	110.5
5.4	114
5.5	117.5
5.6	121
5.7	124.5
5.8	128
5.9	131.5
6	135
6.1	138.5
6.2	142
6.3	145.5
6.4	149
6.5	152.5
6.6	156
6.7	159.5
6.8	163
6.9	166.5
7	170

I throw all of this out because I generally fit the profile you are describing, yet my Exatest showed the contrary indication of low Mg & high Ca which describes insulin resistance. However my A1C does not indicate a high blood sugar problem.

I don't remember how much of the Exatest data you collected in your survey. If you didn't collect the full profile, I would be interested in querying those who've taken the test for the remainder of the data.

BTW, I'm in Lompoc & driving down to Carlsbad, CA tomorrow. My background ectopic count seems lower at sea level than at altitude. I'm not sure what this means.

Thanks for the stimulating reading (as usual)!

**George**

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PC, Referring to adrenergic afibbers you wrote, "Their episodes are triggered by stress, and this translates to a greater assault on their potassium balance than for VMAFers, since the role of aldosterone is greater in adrenergics."

Could you expand on this. How does stress for adrenergic afibbers translate to a greater assault on potassium balance than for vagal afibbers? By assault did you mean reduction of potassium levels? Where does aldosterone come into this? Thanks.

**Bob**

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George, You wrote, "BTW, I'm in Lompoc & driving down to Carlsbad, CA tomorrow. My background ectopic count seems lower at sea level than at altitude. I'm not sure what this means."

Could you be sensitive to the increased iodine in the sea air? That would stimulate your thyroid and make you less vagal.

**Bob**

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George,

I'll address your comments shortly but in the meantime:

#### **Autonomic Balance at High Altitudes**

Danish researchers have found that the parasympathetic branch of the autonomic nervous system takes over and becomes the dominant factor in controlling heart rate and cardiac output when "lowlanders" are exposed to high altitudes (greater than 5000 meters). This could explain why vagal afibbers often experience episodes when mountain climbing or flying in unpressurized airplanes.

*Circulation, Vol. 104, October 9, 2001, pp. 1785-91*

**PC**

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Hi Bob,

Here's an article for you.

*Aldosterone Response To Hypoglycemia: Evidence Of ACTH Mediation*

<http://jcem.endojournals.org/cgi/content/abstract/43/1/173>

This of course is in addition to the effects of insulin and catecholamines directly on blood potassium.

It is important to remember that cortisol is probably the primary stress hormone and that it also binds to aldosterone receptors, although with less affinity than does aldosterone, like about 1/20 the affinity. But there is so much more of it, something in the order of 1000 times more.

Hans has always felt that aldosterone is at the core of LAF. Please read "the play" by Hans Larsen in the Conference Room at <http://www.afibbers.org/conference/session2.pdf>

So, stress of any type (hypoglycemia is only one) results in the release of large amounts of cortisol (in addition to catecholamines). Stress plus hypoglycemia is a particularly potent combination for triggering LAF.

**PC**

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PC, I'm trying to give you constructive criticism. I feel you should respond to my request for clarification yourself, in addition to referring me to other work where I may or may not find the answers after a significant amount of reading. As I mentioned in my original message, my interest was in what you said about potassium and that is what I wanted you to clarify.

**Bob**

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Hi PC,

Well interestingly, my resting HR is a bit lower down at sea level. Bob & I had a bit of discussion about this on the regular board. I've not seen my HR drop (i.e. get more vagal) by going to altitude, however I've not been above 5,000 meters. My normal top is 4,330 m (14,200'). I've only seen my HR drop at altitude when I was under hydrated which brought on altitude sickness. This showed up in a race & I later asked my GP about it. He couldn't answer, but I figured out it was mild altitude sickness.

I think my reduced ectopics are due to the reduced strain on the system at lower altitude.

Generally when people come up to altitude that live at low elevations, their systems work much harder. Because I do it so much, I usually don't even notice, unless I'm above 13,000', however I'm sure my system is working harder. I'm a bit sceptical of the Danish study -- I've just not experienced being more vagal at altitude.

I'm off to enjoy the low-stress So Cal freeways, so enough for now!

**George**

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George, I think you've got the right idea. It's like marathoners who train at high altitude so that their cardiovascular system is stronger when they come down to lower altitudes to compete.

**Bob**

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PC, I read the abstract and the Play that you referred me to and not surprisingly they didn't satisfy my request for clarification that was in my original message.

**Bob**

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Bob,

It sounds like you need to do some reading on cortisol, aldosterone and potassium. It's hard to respond, as I did, if you just don't understand their basic physiology.

**PC**

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Aloha George,

I'd almost forgotten how relaxing those SoCal freeways can be. I'm so jealous (NOT).

Your well informed contrary thoughts are much appreciated.

Hemoglobin A1C is a very poor measure of hypoglycemia, because it reflects mean glycemia over a relatively long period of time. You could have significant swings in blood glucose without effecting HgbA1C. Transient drops would also go undetected with this approach. You can be hyperglycemic for days, spilling the excess glucose into your urine, when blood levels exceed 180 mg/dL. A transient dip in blood glucose due to glutamate or alcohol or skipped meal, etc. would never be detected. The only reason that it has ever been used is because there are so many diabetics running around.

Furthermore, I'm not surprised that you have no suspicion of hypoglycemia being involved in your LAF, since your HDL is low normal.

Also, magnesium deficiency has gained popularity, because it is common in insulin resistance and the prediabetic. But Mg deficiency does not cause insulin resistance. It certainly helps IR along to diabetes, just like I think that it helps trigger LAF. My intracellular Mg was 34.3 (nl range 33.9-41.9) and my intracellular Ca was 5.0 (nl range 3.2-5.0). But these are not measures of insulin resistance. HDL/TG and heart rate recovery are measures of IR/IS.

This then begs the question, "does insulin play a role in your LAF"? Obviously not much. But perhaps that's why you can control your LAF with electrolyte balance and the rest of have/had to struggle.

Clearly, there is more to LAF than insulin sensitivity. Even among the physically fit with increased IS (high HDL/TG) not everyone gets LAF. I have no idea what else is contributing to expression of this arrhythmia in some but not others.

But there are other factors to consider wrt potassium balance, e.g., thyroid disease, adrenal disease and renal disease. All can contribute to urinary potassium wasting.

However, the results of LAFS-11 suggest that insulin sensitivity and occasional potassium shortfall are key players. I can see no other way to interpret the data.

**PC**

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Hi PC,

I appreciate the effort you have put into studying the relationship between insulin resistance/sensitivity and LAF. The above information and links is most helpful.

For years I have suspected that I am hypoglycemic but the tests always come back negative. Maybe this deserves more attention.

I am currently leaning towards more of an endocrinological explanation for LAF, but it's a hugely intertwined/interconnected approach. I will have to brush up on my biochemistry and physiology!

**Mark**

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Aloha Mark,

Thank you for your comments. I enjoy pursuing the story behind this affliction almost as much as I didn't enjoy having it.

I'm hoping Hans will allow me one more opportunity to access the participants of the BB, CR and afibbers.org. I think the more objective the data is the less easily LAFers will be overlooked by the medical mainstream as a separate and distinct entity.

Hypoglycemia is exceedingly hard to diagnose in general and its transient role in an LAF episode even more so.

But holiday heart is fairly common. I know of several people that have encountered it. I'm sure it is often a onetime thing and victims are rarely aware of what actually happened.

I know of several others that have experienced AF as a result of low potassium secondary to its loss in urine during diuretic therapy for hypertension.

Potassium balance is the key to its control, at least initially before episodes can cause atrial enlargement.

LAFers are a subset that for some reason struggle even more with blood fluctuations of this electrolyte. It's



undoubtedly a mixed bag that will slowly reveal itself as more layers are removed.

**PC**

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Hi PC,

My neighbor, the internist, says that almost all of the cases of AF that he sees are holiday heart.

If you go for another round of questions, I've thought of going through Peggy's "The List" and contacting those people, 1) for an update, and 2) to gather anthropometric and other data to see if it can be determined what is different about this group and 3) to see if it appears to be one or multiple discreet populations. If I pursue this quest, I'd love some help figuring out what to ask.

As to hypoglycemia, I guess I have a hard time having this affliction divided into subcategories - one that is a precursor to type II diabetes and another that is an oversensitivity to insulin. I would tend to lump all into the first category.

**George**

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Hi George,

I think that's an excellent suggestion, one that has occurred to me as well. I'm sure we could come up with some good questions. We can run it by Hans upon his return, unless Peggy is able to provide a list. We can get their email addresses from previous posts.

Regarding hypoglycemia, I can understand your misgivings.

Hypoglycemia can be caused by a number of different mechanisms. In fact the most common is unrelated to either one you mentioned. "Iatrogenic" hypoglycemia due to excess exogenous insulin, i.e., too much prescribed or taken by mistake. Insulin is like coumadin in this regard. How much we need is a moving target.

Prediabetic/diabetic hypoglycemia is in part related to magnesium deficiency. It's caused by loss of glucose in the urine and subsequent insufficient reservoir of glucose (glycogen). They can become hypoglycemic usually hours later. Such individuals are insulin resistant. The insulin is always increased but the receptor sites are not responding.

So, too much exogenous (subcutaneous) insulin or too little insulin effect (insulin resistance) can cause hypoglycemia. One is immediate and the other is delayed. However, in neither event does endogenous insulin ever directly cause hypoglycemia in this prediabetic/diabetic crowd.

Idiopathic postprandial hypoglycemia or reactive hypoglycemia is unrelated to diabetes or prediabetes. That is the kind of hypoglycemia of which I'm talking. In these individuals insulin levels are low, because their receptor sites are much more sensitive to insulin. For them the hypoglycemia is gradual and prolonged.

And remember that I am using the term hypoglycemia to include prolonged low range of normal blood glucose level.

Diabetic physiology is complex enough without including postprandial or reactive hypoglycemia. Plenty of physicians don't understand it. As a lab director, I can't tell you how many physicians I had to call about misguided attempts to evaluate reactive hypoglycemia via 5 hour GTT.

**PC**

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Hi PC,

Thanks for educating me in my stubborn (mule) mode.

I don't really count iatrogenic hypoglycemia in the AF equation - though it is certainly a big problem for diabetics. In fact

it is the one that can kill someone.

As to the questions, I'd probably just have to search "The List" and gather all the email addresses.

BTW, I'm enjoying So Cal. It is great when I'm not on the freeways. Beach life does have its attractions.

## **George**

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The bulletin board and the conference room are converging once more, and this time i want to import something from the regular bb into this forum discussion, contrary to how i was feeling about the nattokinase discussion, which at the time i thought should be more on the regular bb so it could be found via the search function. Since then i have reconsidered that feeling, and at least in this case i think all that bb material ought to be concentrated in one spot, here in the CR.

Therefore here is a link to that bb discussion, started by a classic Jackie post, encyclopedic and thought-provoking:

[Jackie 2/8/06, 18:17] [http://www.afibbers.net/forum/read.php?f=4&i=1391&t=1391#reply\\_1391](http://www.afibbers.net/forum/read.php?f=4&i=1391&t=1391#reply_1391)

In the latter part of that Jackie article on the vagus nerve, she gets into the influence of electrolyte balance on body functions dependent on the vagus nerve. Some excerpts from an article she quotes:

"... influence of calcium and potassium on the function of the autonomic nervous system. In particular, I pointed out that potassium deficiencies produce a weakened parasympathetic response..."

"... calcium acts much the same as stimulation of the sympathetic nerves while potassium and sodium produce a parasympathetic (vagus) effect..."

"... normal control of the heart muscle depends upon a certain equilibrium between the action of the sympathetic and the vagus nerves supplying the heart and a relative proportion between the ions, particularly the calcium and potassium inside the cells. A relative increase in calcium over potassium produced a sympathetic effect (increased rate and force of contraction). Conversely, a relative increase in potassium over calcium increased parasympathetic action..."

[This last bears on why calcium supplements give some people afib].

And here is the tie-in with the topic of insulin and afib:

"... Potassium is required for the assimilation of simple sugars and the greater the consumption of mineral deficient simple (refined) sugars, the more likely a potassium deficiency exists. In fact, one of the major causes of potassium deficiencies in North America is excessive sugar consumption and not diuretic use, as many suppose.

Sympathetic dominance can occur by default when potassium deficiencies occur and vagus innervation to the tissues becomes inadequate..."

This last bears on why intake of a large double chocolate muffin, for one personal example, gives me that fast, hard, uncomfortable heartbeat which is completely quelled by taking K gluconate in water. As PC has explained elsewhere, insulin depletes potassium stores.

The article Jackie is quoting there is this one:

<http://www.worldchiropracticalliance.org/tcj/1998/dec/dec1998loomis.htm>

Please do read it, it is full of afib-important stuff.

## **PeggyM**

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This statement from Peggy's post describes exactly what I did to end my afib:

"... Potassium is required for the assimilation of simple sugars and the greater the consumption of mineral deficient simple (refined) sugars, the more likely a potassium deficiency exists. In fact, one of the major causes of potassium deficiencies in North America is excessive sugar consumption and not diuretic use, as many suppose."

After my last LAF attack in Feb 2003 I spent 5 days in the cardio ward having every test under the sun and was released with "you have LAF".

This is when I scrutinised my diet and was shocked to find the amount of sugar I consumed. I then went on a very strict diabetic diet. To this day, I have not eaten a piece of chocolate, lollies or birthday cake (much to my families and friends disgust) as well as all obvious and hidden sources of sugar. I scrutinise everything for sugar with the one exception..

Alcohol. I still drink a moderate amount of beer and red wine but in steady amounts, not binge drinking (you have to have some vices). In one week I will be celebrating 3 years afib free and one and half months totally ectopic free. At this point in time I am a normal person again.

Why hasn't this potassium and excess sugar link been discussed before? This fills in a very large chunk of the AF jigsaw for me. I have always been wondering if cutting out the sugar was the real reason for being afib free for 3 years.

Please discuss.

**Dean**

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Dean, i have spent the last few days rummaging around in *The Chiropractic Journal* in general, and Dr. Loomis's columns in particular. Here is the url for that journal's archives:

<http://www.worldchiropracticalliance.org/tcj/archives.htm>

I should say here that i have never been to a chiropractor and know nothing whatever about chiropractic except that i cannot afford it. So the parts of these columns, which concern manipulations and the like, are quite lost on me.

However, Dr. Loomis talks a lot about sugar causing K deficiency, and about sympathetic/parasympathetic dominance, among other interesting topics. He has a commendable interest in nutrition as it affects body functions, though he always gets around to a plug for the line of digestive enzymes he promotes. I have no idea whether these might be of value, but Jackie would know about them. The language he uses about them strongly resembles the same things Jackie has said in posts about digestive enzymes.

From a column called "Potassium deficiency", Nov. '98

<http://www.worldchiropracticalliance.org/tcj/1998/nov/nov1998loomis.htm>

"The autonomic nervous system is one of two control systems the body uses to control homeostasis, the other being the endocrine system...

... Nutrition plays a key role by enabling a cell/tissue/organ to respond to autonomic stimulation....

... In order to respond to parasympathetic stimulation it is necessary for potassium to accumulate inside the cells of those tissues being stimulated. In order for tissues to respond to sympathetic stimulation, calcium must accumulate inside the cells.... In other words, a calcium deficiency results in symptoms of parasympathetic dominance because the cells lack adequate calcium to respond to sympathetic stimulation. A potassium deficiency results in symptoms of sympathetic dominance because the cells lack adequate potassium to respond to parasympathetic stimulation....

... The symptoms of sympathetic dominance are many, and I refer you to the many good textbooks in your library for details. More germane to this month's column are the many symptoms of potassium deficiency, primarily constipation, stiff or sore joints, and bradycardia (or the feeling of skipped heart beats).

A "clouded sensorium" is another symptom of potassium deficiency, one being seen more and more in our present society. It is usually referred to as the inability to think clearly or concentrate (attention deficient). Patients may even

speak of a feeling of separation of their mind from their body..."

This article was very interesting to me, and i do recommend reading it in full. I have just excerpted a few sentences here. Dr. Loomis repeats this paragraph in all of his columns on this subject:

"When asked to identify the major cause(s) of potassium deficiencies in North America, many respond by blaming diuretics and blood pressure medications. But, that is seldom the case. The primary cause of potassium depletion is excessive sugar use, a very common condition indeed."

Personally i would also blame low intake of K in the SAD diet, but surely the ordinary large sugar and simple carbohydrate intake rapidly depletes whatever small amount is eaten.

I am beginning to get a better understanding why paleodiet was so immediately helpful to me. Not only did it eliminate all added msg and most sources of free glutamate, it also both increased the amount of K i got in food, and sharply reduced high glycemic load foods.

While i was at it i read all of Dr. Loomis's columns on this subject. In case others may find them interesting, here are links to them:

<http://www.worldchiropracticalliance.org/tcj/1997/jan/jan1997loomis.htm>  
[The sympathetic-parasympathetic balance]

<http://www.worldchiropracticalliance.org/tcj/1998/dec/dec1998loomis.htm>

<http://www.worldchiropracticalliance.org/tcj/1999/jun/jun1999loomis.htm>

<http://www.worldchiropracticalliance.org/tcj/1999/mar/mar1999loomis.htm>

<http://www.worldchiropracticalliance.org/tcj/1999/aug/aug1999loomis.htm>

<http://www.worldchiropracticalliance.org/tcj/2002/mar/mar2002loomis.htm>

<http://www.worldchiropracticalliance.org/tcj/2003/apr/apr2003loomis.htm>

<http://www.worldchiropracticalliance.org/tcj/2004/jan/loomis.htm>

<http://www.worldchiropracticalliance.org/tcj/2004/mar/loomis.htm>

### ***PeggyM***

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Hi Peggy,

There is another website afibbers have discussed and that is the Acu-Cell website. This is also edited by a chiropractor and explains similar relationships between the vagus nerve/potassium and spinal manipulation. It seems chiropractors are very educated on this subject:

<http://www.acu-cell.com/znk.html>

I find the excess sugar and low K link very interesting in regards to the paleo diet. Fran was the one who went all out on a strict paleo and cured her afib. Fran always said it was the glutamate in her diet causing afib but was it actually excess sugar? If I remember rightly, didn't Fran have a sweet tooth before starting the paleo?

PC surmises in the current newsletter that af could be "the opposite of diabetes". So in effect could excess sugar be behind af as is excess sugar in diabetes? Fran cut out sugar, I cut out sugar and you and several others on the paleo have had great success. Cutting out sugar is the one thing in common. Wonder what PC has to say?

### ***Dean***

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Dean,

If I remember right, you are also taking a PPI, correct? If so, I wonder if your good results would be the same without it.

**Marian**

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Hi Marian,

I still take 20mg Losec a day. I tried to cut it down to 10mg but ectopics came back. Same with the natto food- tried to cut down to 4 times a week but ectopics returned. At the moment I operate in a very narrow band to stay ectopic free:

- 20mg Losec a day
- natto no less than 5 days week
- cut out all sugar

If I vary from this the ectopics come back.

It's interesting that "Michael in San Francisco" is in the same narrow band as I am with his supplement regime. He strayed slightly from his regime and had a breakthrough afib attack after being 3yrs afib free.

**Dean**

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Hi Dean,

Sorry to be so tardy in responding, but I've been having some family related health problems.

You're preaching to the choir wrt dietary sugar and LAF.

I think the medical community may be just beginning to pick up on the sugar connection with AF. Unfortunately they are all looking at it from an insulin resistance standpoint. LAF is the unwanted stepchild. Clinicians neither know what to make of it nor how to treat it.

I finally submitted something to Lancet on just this topic.

**PC**

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Peggy,

From your post above:

*"In other words, a calcium deficiency results in symptoms of parasympathetic dominance because the cells lack adequate calcium to respond to sympathetic stimulation. A potassium deficiency results in symptoms of sympathetic dominance because the cells lack adequate potassium to respond to parasympathetic stimulation...."*

Background of relevance here: my last intracellular (rbc) Ca was right at bottom of range - as was Mag (leucocyte). My AF to date has all occurred at 3am time and as such can broadly be regarded as vagal. My blood sugar is always around 5.4 and as such is low.

From your quote above, my low Ca could explain my vagal predominance. However, if a K deficiency results in a sympathetic dominance, then that would surely rule out K shortage as a cause for LAF? As such, surely the adrenergic folks should respond far more favourably to K supplementation than VMAFs?? But then we both know that George and other VMAFs here respond well to K supplementation. Perhaps I'm just over-simplifying ONE single aspect of what is a highly complex picture... as usual!

Cheers,

**Mike F.**

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Mike, if i am understanding correctly, vagal afibbers are too far to the sympathetic side, and adrenergic afibbers are too far to the parasympathetic side. If K deficiency makes for sympathetic dominance, and K intake pushes vagal afibbers more toward the middle where they belong, then that could be why vagal afibbers are helped by potassium. Makes sense to me.

Mike, where you been? Have noticed your conspicuous absence from the forum.

### ***PeggyM***

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Mike et al, i think my previous post was in error. While browsing around in IHN looking for information on the ANS, i found an article containing the following quote:

"... - excessive adrenergic (sympathetic) and inadequate vagal (parasympathetic) response –... "

<http://www.yourhealthbase.com/database/a112f.htm>

so it looks like i had it exactly backwards. Sorry, i am a bit slow but i do eventually catch on. And i am completely unable to explain what you wanted explained, Mike, and am as far at sea on it as you. PC, can you straighten this out?

### ***PeggyM***

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Aloha Peggy,

As always, thank you for your input.

VMAFers (vagally mediated AF) have excessive vagal tone. Conventional thinking on adrenergic LAF is that they may have excessive sympathetic tone. IMHO this is not so and is what differentiates true LAF from pathologic AF.

Pathologic AF is associated with a constant increase in sympathetic tone, whereas for ALAF this may be only transient. The actual transition from sympathetic back to parasympathetic may be the culprit. Much has been written on this latter point.

I think it is important to separate that which causes the PACs from that which causes the fertile soil for receiving those PACs and sustaining AF. Potassium imbalance contributes to the former predominantly and autonomic tone contributes to the latter predominantly, but there's lots of overlap.

I personally believe that autonomic tone is for the most part independent of any change in blood potassium and vice versa. Any shortfall in blood potassium is bad for either type of LAF.

There are too many reflexive neurohormonal mechanisms at work to equate either arm of the autonomic nervous system with blood potassium level alone.

### ***PC***

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PC, i can do pretty well with that answer until i come to this part:

"I personally believe that autonomic tone is for the most part independent of any change in blood potassium and vice versa. Any shortfall in blood potassium is bad for either type of LAF. There are too many reflexive neurohormonal mechanisms at work to equate either arm of the autonomic nervous system with blood potassium level alone."

Can you elaborate on that a little more?

### ***PeggyM***

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Hi Peggy,

Autonomic tone is controlled by many things independent of blood potassium, e.g., genes, weight, fitness level. I don't pretend to know all about the physiology of this, except to say that it appears to be primarily a CNS (central nervous system) thing.

Potassium can indirectly impact one measure of autonomic tone (blood pressure) via blood Na/K ratio. When this goes down (increased blood K), this is sensed directly by the adrenal cortex and aldosterone is secreted. This results in the retention of Na and water and an increase in blood volume. But then this increase in blood volume results in greater hydrostatic pressure sensed by renal baroreceptors and RAS (renin angiotensin system) activity decreases to counter this. The overall result is good for both subtypes of LAF. So, on a superficial level increased blood potassium lessens angiotensin, a potent vasoconstrictor, and blood pressure. One might call this sympatholytic, but I for one would not, because autonomic tone is primarily centrally controlled, even though it works through peripheral organs like the adrenal gland and various baroreceptors.

Hope this is not too technical.

**PC**

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